chemoattraction of microglia cells. The anti-TGF-β treatment would induce the opposite effects. Inhibition of TGF-β activity leads to numerous non-specific cellular responses, which may even lead to unwanted side effects. One object of the invention is to avoid such potential unwanted side effects.

Please substitute the following paragraph for page 3, paragraph 4.

--According to the invention, there can further be used those inhibitor substances which are selected from the group consisting of N-oxaloglycine; Zn salts; pyridine derivatives, such as 5-arylcarbonyamino- or 5-arylcarbamoyl- derivatives, 2-carboxylate, 2,5 dicarboxylate, their ethyl esters or ethyl amides or -5-acyl sulfonamides, 2,4 dicarboxylate, their ethyl esters or ethylamides, or dimethoxyethylamides; 3,4 bipyridine, such as 5-amino-6-(1H)-one, 1,6-dihydro-2-methyl-6-oxo-5-carbonitril; 2,2'-bipyridine, such as 5,5'-dicarboxylic acid or its pharmaceutically acceptable salts, 4,4'-dicarboxylic acid ethyl ester or ethyl amide; 3,4'-dihydroxybenzoate, such as the diethyl ester; proline and its structural and functional analoges; beta -aminopropionitrile; desferrioxamine; anthracyclines; 2,7,8-trihydroxy anthraquinones, fibrostatin-C; coumalic acid or its pharmaceutically acceptable salts; 5-oxaproline, beta -lactam antibiotics.--

Please substitute the following paragraph for page 6, paragraph 3.

--The mechanically transected postcommissural fornix of the adult rat, a unidirectional and well-characterized fiber tract (8,9), was used to determine whether specific biochemical or immunochemical modulation of BM formation would provide a means to stimulate axon regeneration. Here we report that lesion-induced BM deposition can be significantly reduced by local injection of anti-collagen IV antibodies or alpha, alpha dipyridyl, an inhibitor of collagen

triple helix formation and synthesis. Reducing the collagen network allowed massive axon elongation across the lesion site. The regenerating fornix fibers followed the original pathway, reinnervated their appropriate target, the mammillary body, were remyelinated and attained nearly normal conduction properties. On failure of adult mammalian CNS axons, we examined the spatio-temporal distribution pattern after penetrant CNS lesion and determined whether remodelling allows structural and functional regeneration of a transected CNS fiber tract.

Please substitute the following paragraph for the paragraph bridging pages 9 and 10.

Further preferred embodiments for restitution of functional circuitry after traumatic CNS lesion are the remyelination of regenerated fibers, the re-establishment of synaptic connections with the appropriate target and the restoration of normal conduction properties. Structural and functional properties of the regenerating axons were investigated using immunohistochemical, morphological and electrophysiological methods. Immunohistochemistry with an antibody against myelin basic protein demonstrated the remyelination of regenerated fornix axons along their entire length as early as 4 weeks after surgery (data not shown). This observation was confirmed by ultrastructural analysis of anterogradely WGA-HRP labeled axons in the distal stump which showed clear evidence of compact myelin sheath formation (Fig. 3c). In addition, ultrastructural studies provided evidence for the reestablishment of synaptic connections of regenerating axons within the mammillary body. Tracer reaction product was identified in presynaptic profiles with round vesicles that formed asymmetric synaptic junctions at unlabeled dendrites (Fig. 3d, e). The ultrastructural features of the labeled presynaptic profiles correspond to those described for the RA-type (round, asymmetric) of synaptic terminal, which is considered to be of subicular origin (8). The electrophysiological properties of regenerated fibers were studied using extracellular in vitro recording techniques

applied to sagittal brain slices (400 mu m) of 8 unlesioned rats and 4 treated animals showing regenerated fiber tracts. In unlesioned animals electrical stimulation of the fornix fibers elicited an extracellular action potential with an amplitude of 1.02 +- 0.14 mV and a conduction velocity of 0.48 +- 0.05 m/s (mean +- SEM, n=16, Fig. 4b-d). This axonal conduction velocity corresponds well to previously reported measurements (about 0.5 m/s for hippocampal Schaffer collaterals (15). Similar values for action potential amplitude and conduction velocity (1.12 +/- 0.21 mV, 0.46 +/- 0.1 m/s, n=5) were obtained in axon regenerating animals when the stimulating (S) and the recording (R) electrodes were positioned proximally to the lesion site (see S1 and R1 in Fig. 4a). In the latter animals, functionally intact fibers showing normal extracellular action potential amplitude and conduction velocity could also be demonstrated across (S3 and R3 in Fig. 4a; 0.8 +/- 0.29 mV, 0.54 +/- 0.14 m/s, n=3) and distal to the lesion site (S2 and R2 in Fig. 4a; 0.91 +/- 0.24 mV, 0.43 +/- 0.06 m/s, n=4) (Fig. 4c, d). In all animals, the stimulus-evoked extracellular responses were blocked by Tetrodotoxin, confirming their nature as Na+-dependent action potentials (Fig. 4b). From these data we conclude that the reorganization of the fornix tract is accompanied by structural and functional recovery of the regenerated axons.

Please substitute the following paragraph for page 12, paragraph 2.

Electrophysiology and biocytin injections. Sagittal slices of 400 m thickness were cut on a vibratome and maintained at 34-35 °C in an interface-type recording chamber. Artificial cerebrospinal fluid (ACSF) consisted of (in mM) 124 NaCl, 3 KCl, 1.25 NaH_2PO_4, 1.8 $MgSO_4$, 1.6 $CaCl_2$, 26 $NaHCO_3$ and 10 glucose with a pH of 7.4 when saturated with 95% O_2 - 5% CO_2. Stimuli of 100 µs, 5-20 V were delivered via a bipolar tungsten electrode. Extracellular action potentials were registered with a recording electrode (3-5 MW) located in the middle of the postcommissural

fornix. Tetrodotoxin (TTX, Sigma) was applied locally in a concentration of 10 μM (dissolved in ACSF) with a broken micropipette placed on the slice surface near the recording site. Injections of a small biocytin (Sigma) crystal into the fornix were performed with a miniature needle. After an incubation period of 8-10 h in the interface chamber, slices were fixed in 4 % paraformaldehyde, resectioned and reacted with ABC peroxidase reagent (Vector Labs). --

IN THE CLAIMS

Cancel claims 1-17, without prejudice or disclaimer.

Add the following claims.

18. A method for the improvement of neuronal regeneration comprising specific inhibition of basal membrane formation induced by a lesion of neuronal tissue.

19. The method according to claim 18, wherein the formation of the basal membrane is inhibited by applying an inhibitor substance, wherein the inhibitor substance is an inhibitor of the synthesis of basal membrane building elements, an inhibitor of the assembly of basal membrane building elements, or the inhibitor of the synthesis of basal membrane building elements and the inhibitor of the assembly of basal membrane building elements to a body in need thereof.

20. The method of claim 19, wherein the basal membrane building elements are collagen IV, laminin, entactin, accessory substances for proper function or assembly of the basal